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=> s FEN-1 and (dna or gene or nucleic acid or rna or polynucleotide) and 1990-1995/py

   2 FILES SEARCHED...
   4 FILES SEARCHED...
L1          39 FEN-1 AND (DNA OR GENE OR NUCLEIC ACID OR RNA OR POLYNUCLEOTIDE)
               AND 1990-1995/PY

=> dup rem l1

PROCESSING COMPLETED FOR L1
L2          10 DUP REM L1 (29 DUPLICATES REMOVED)

=> s l2 and 5'-flap and (endonuclease or endonucleolytic)

MISMATCHED QUOTE 'AND 5'-FLAP'
Quotation marks (or apostrophes) must be used in pairs,
one before and one after the expression you are setting
off or masking.

=> s l2 and 5-flap and (endonuclease or endonucleolytic)

L3          1 L2 AND 5-FLAP AND (ENDONUCLEASE OR ENDONUCLEOLYTIC)

=> d l3 ibib ab


L3   ANSWER 1 OF 1  MEDLINE
ACCESSION NUMBER:   95181442     MEDLINE
DOCUMENT NUMBER:    95181442
TITLE:              DNA structural elements required for FEN
                    -1 binding.
AUTHOR:             Harrington J J; Lieber M R
CORPORATE SOURCE:   Department of Pathology, Stanford University School of
                    Medicine, California 94305..
SOURCE:             JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Mar 3) 270
```

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, **(1995 Mar 3)** 270 (9) 4503-8.
Journal code: HIV. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199506

AB In eukaryotic cells, a **5'-flap DNA endonuclease** and a double-stranded **DNA** 5'-exonuclease activity reside within a 42-kDa enzyme called **FEN-1** (flap **endonuclease**-1 and 5(five)'-exonuclease). This endo/exonuclease has been shown to be highly homologous to human XP-G, Saccharomyces cerevisiae RAD2, and S. cerevisiae YKL510. Like **FEN-1**, these related structure-specific nucleases recognize and cleave a branched **DNA** structure called a **DNA** flap and its derivative, called a pseudo Y-structure. To dissect the important structural components of the **DNA** flap structure, we have developed a mobility shift assay. We find that the Fadj strand (located adjacent to the displaced flap strand) is necessary for efficient binding and cleavage of flap structures by **FEN-1**. When this strand is absent or when it is present, but recessed from the elbow of the flap strand, binding efficiency drops. Further investigation of the role of the Fadj strand using double flap structures reveals that the Fadj strand is necessary to provide a double-stranded template upon which **FEN-1** can bind near the elbow of the flap strand. These results provide a basis for understanding how this structure-specific nuclease recognizes a variety of **DNA** substrates.

=> d his

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FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS, SCISEARCH, BIOTECHDS' ENTERED AT 14:03:50 ON 01 MAR 2001

L1 39 S FEN-1 AND (DNA OR GENE OR NUCLEIC ACID OR RNA OR POLYNUCLEOTI
L2 10 DUP REM L1 (29 DUPLICATES REMOVED)
L3 1 S L2 AND 5-FLAP AND (ENDONUCLEASE OR ENDONUCLEOLYTIC)

=> s l2 and mobility shift assay

L4 1 L2 AND MOBILITY SHIFT ASSAY

=> s l2 and double flap

L5 1 L2 AND DOUBLE FLAP

=> s l2 and (cleaving polynucleotide or cleaving nucleic acid or cleaving dna)

L6 0 L2 AND (CLEAVING POLYNUCLEOTIDE OR CLEAVING NUCLEIC ACID OR CLEAVING DNA)

=> s l2 and (cleaving a polynucleotide or cleaving a nucleic acid or cleaving a dna)

2 FILES SEARCHED...
L7 0 L2 AND (CLEAVING A POLYNUCLEOTIDE OR CLEAVING A NUCLEIC ACID OR CLEAVING A DNA)

```
=> s l2 and (3-dna or 5-dna)

L8            1 L2 AND (3-DNA OR 5-DNA)

=> s l2 and (3-dna or 5-dna)

   4 FILES SEARCHED...
L9            1 L2 AND (3-DNA OR 5-DNA)

=> d l9


L9   ANSWER 1 OF 1  MEDLINE
AN   95293376     MEDLINE
DN   95293376
TI   Sequence of human FEN-1, a structure-specific
     endonuclease, and chromosomal localization of the gene (FEN1) in
     mouse and human.
AU   Hiraoka L R; Harrington J J; Gerhard D S; Lieber M R; Hsieh C L
CS   Department of Pathology, Stanford University School of Medicine,
     California 94305-5324, USA.
NC   5T32CA09302 (NCI)
     T32CA09151 (NCI)
SO   GENOMICS, (1995 Jan 1) 25 (1) 220-5.
     Journal code: GEN. ISSN: 0888-7543.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
OS   GENBANK-L37374
EM   199509


=> d his

     (FILE 'HOME' ENTERED AT 14:02:59 ON 01 MAR 2001)

     FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS, SCISEARCH, BIOTECHDS' ENTERED AT
     14:03:50 ON 01 MAR 2001
L1           39 S FEN-1 AND (DNA OR GENE OR NUCLEIC ACID OR RNA OR POLYNUCLEOTI
L2           10 DUP REM L1 (29 DUPLICATES REMOVED)
L3            1 S L2 AND 5-FLAP AND (ENDONUCLEASE OR ENDONUCLEOLYTIC)
L4            1 S L2 AND MOBILITY SHIFT ASSAY
L5            1 S L2 AND DOUBLE FLAP
L6            0 S L2 AND (CLEAVING POLYNUCLEOTIDE OR CLEAVING NUCLEIC ACID OR C
L7            0 S L2 AND (CLEAVING A POLYNUCLEOTIDE OR CLEAVING A NUCLEIC ACID
L8            1 S L2 AND (3-DNA OR 5-DNA)
L9            1 S L2 AND (3-DNA OR 5-DNA)

=> d l2 1-10 ibib ab


L2   ANSWER 1 OF 10  MEDLINE                                  DUPLICATE 1
ACCESSION NUMBER:    96107187     MEDLINE
DOCUMENT NUMBER:     96107187
TITLE:               Calf 5' to 3' exo/endonuclease must slide from a 5' end of
                     the substrate to perform structure-specific cleavage.
AUTHOR:              Murante R S; Rust L; Bambara R A
```

AUTHOR: Murante R S; Rust L; Bambara R A
CORPORATE SOURCE: Department of Biochemistry, University of Rochester School of Medicine and Dentistry, New York 14642, USA.
CONTRACT NUMBER: GM24441 (NIGMS)
T32-GM07102 (NIGMS)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, **(1995 Dec 22)** 270 (51) 30377-83.
Journal code: HIV. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199604

AB Calf 5' to 3' exo/endonuclease, the counterpart of the human **FEN -1** and yeast RTH-1 nucleases, performs structure-specific cleavage of both **RNA** and **DNA** and is implicated in Okazaki fragment processing and **DNA** repair. The substrate for endonuclease activity is a primer annealed to a template but with a 5' unannealed tail. The results presented here demonstrate that the nuclease must enter the 5' end of the unannealed tail and then slide to the region of hybridization where the cleavage occurs. The presence of bound protein or a primer at any point on the single-stranded tail prevents cleavage. However, biotinylation of a nucleotide at the 5' end or internal to the tail does not prevent cleavage. The sliding process is bidirectional. If the nuclease slides onto the tail, later binding of a primer to the tail traps the nuclease between the primer binding site and the cleavage site, preventing the nuclease from departing from the 5' end. A model for 5' entry, sliding, and cleavage is presented. The possible role of this unusual mechanism in Okazaki fragment processing, **DNA** repair, and protection of the replication fork from inappropriate endonucleolytic cleavage is presented.

L2 ANSWER 2 OF 10 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 95403394 MEDLINE
DOCUMENT NUMBER: 95403394
TITLE: Lagging strand **DNA** synthesis at the eukaryotic replication fork involves binding and stimulation of **FEN-1** by proliferating cell nuclear antigen.
AUTHOR: Li X; Li J; Harrington J; Lieber M R; Burgers P M
CORPORATE SOURCE: Department of Biochemistry, Washington University School of Medicine, St. Louis, Missouri 63110, USA..
CONTRACT NUMBER: GM32431 (NIGMS)
CA51105 (NCI)
GM43236 (NIGMS)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, **(1995 Sep 22)** 270 (38) 22109-12.
Journal code: HIV. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199512

AB The 5'-->3'-exonuclease domain of Escherichia coli **DNA** polymerase I is required for the completion of lagging strand **DNA** synthesis, and yet this domain is not present in any of the eukaryotic **DNA** polymerases. Recently, the **gene** encoding the functional and evolutionary equivalent of this 5'-->3'-exonuclease domain has been identified. It is called **FEN-1** in mouse and human cells and RTH1 in Saccharomyces cerevisiae. This 42-kDa enzyme is

human cells and RTH1 in Saccharomyces cerevisiae. This 42-kDa enzyme is required for Okazaki fragment processing. Here we report that **FEN-1** physically interacts with proliferating cell nuclear antigen (PCNA), the processivity factor for **DNA** polymerases delta and epsilon. Through protein-protein interactions, PCNA focuses **FEN-1** on branched **DNA** substrates (flap structures) and on nicked **DNA** substrates, thereby stimulating its activity 10-50-fold but only if PCNA can functionally assemble as a toroidal trimer around the **DNA**. This interaction is important in the physical orchestration of lagging strand synthesis and may have implications for how PCNA stimulates other members of the **FEN-1** nuclease family in a broad range of **DNA** metabolic transactions.

L2 ANSWER 3 OF 10 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 95181442 MEDLINE
DOCUMENT NUMBER: 95181442
TITLE: **DNA** structural elements required for **FEN-1** binding.
AUTHOR: Harrington J J; Lieber M R
CORPORATE SOURCE: Department of Pathology, Stanford University School of Medicine, California 94305..
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, **(1995 Mar 3)** 270 (9) 4503-8.
Journal code: HIV. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199506
AB In eukaryotic cells, a 5'-flap **DNA** endonuclease and a double-stranded **DNA** 5'-exonuclease activity reside within a 42-kDa enzyme called **FEN-1** (flap endonuclease-1 and 5(five)'-exonuclease). This endo/exonuclease has been shown to be highly homologous to human XP-G, Saccharomyces cerevisiae RAD2, and S. cerevisiae YKL510. Like **FEN-1**, these related structure-specific nucleases recognize and cleave a branched **DNA** structure called a **DNA** flap and its derivative, called a pseudo Y-structure. To dissect the important structural components of the **DNA** flap structure, we have developed a mobility shift assay. We find that the Fadj strand (located adjacent to the displaced flap strand) is necessary for efficient binding and cleavage of flap structures by **FEN-1**. When this strand is absent or when it is present, but recessed from the elbow of the flap strand, binding efficiency drops. Further investigation of the role of the Fadj strand using double flap structures reveals that the Fadj strand is necessary to provide a double-stranded template upon which **FEN-1** can bind near the elbow of the flap strand. These results provide a basis for understanding how this structure-specific nuclease recognizes a variety of **DNA** substrates.

L2 ANSWER 4 OF 10 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 95349595 MEDLINE
DOCUMENT NUMBER: 95349595
TITLE: Mutations in RAD27 define a potential link between G1 cyclins and **DNA** replication.
AUTHOR: Vallen E A; Cross F R
CORPORATE SOURCE: Rockefeller University, New York, New York 10021-6399, USA..
CONTRACT NUMBER: GM7238 (NIGMS)
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, **(1995 Aug)** 15 (8)

SOURCE: MOLECULAR AND CELLULAR BIOLOGY, **(1995 Aug)** 15 (8) 4291-302.
Journal code: NGY. ISSN: 0270-7306.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199511

AB The yeast Saccharomyces cerevisiae has three G1 cyclin (CLN) genes with overlapping functions. To analyze the functions of the various CLN genes, we examined mutations that result in lethality in conjunction with loss of cln1 and cln2. We have isolated alleles of RAD27/ERC11/YKL510, the yeast homolog of the **gene** encoding flap endonuclease 1, **FEN-1**.cln1 cln2 rad27/erc11 cells arrest in S phase; this cell cycle arrest is suppressed by the expression of CLN1 or CLN2 but not by that of CLN3 or the hyperactive CLN3-2. rad27/erc11 mutants are also defective in **DNA** damage repair, as determined by their increased sensitivity to a **DNA**-damaging agent, increased mitotic recombination rates, and increased spontaneous mutation rates. Unlike the block in cell cycle progression, these phenotypes are not suppressed by CLN1 or CLN2. CLN1 and CLN2 may activate an RAD27/ERC11-independent pathway specific for **DNA** synthesis that CLN3 is incapable of activating. Alternatively, CLN1 and CLN2 may be capable of overriding a checkpoint response which otherwise causes cln1 cln2 rad27/erc11 cells to arrest. These results imply that CLN1 and CLN2 have a role in the regulation of **DNA** replication. Consistent with this, GAL-CLN1 expression in checkpoint-deficient, mec1-1 mutant cells results in both cell death and increased chromosome loss among survivors, suggesting that CLN1 overexpression either activates defective **DNA** replication or leads to insensitivity to **DNA** damage.

L2 ANSWER 5 OF 10 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1995:326612 BIOSIS
DOCUMENT NUMBER: PREV199598340912
TITLE: Genetic organization of human **FEN-1**.
AUTHOR(S): Shen, Binghui; Gonzales, Julia A.; Marrone, Babetta L.; Park, Min S.
CORPORATE SOURCE: Life Sciences Div., Los Alamos National Lab., Los Alamos, NM 87545 USA
SOURCE: Journal of Cellular Biochemistry Supplement, (1995) Vol. 0, No. 21A, pp. 303.
Meeting Info.: Keystone Symposium on Repair and Processing of DNA Damage Taos, New Mexico, USA March 23-29, 1995
ISSN: 0733-1959.
DOCUMENT TYPE: Conference
LANGUAGE: English

L2 ANSWER 6 OF 10 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 95293376 MEDLINE
DOCUMENT NUMBER: 95293376
TITLE: Sequence of human **FEN-1**, a structure-specific endonuclease, and chromosomal localization of the **gene** (FEN1) in mouse and human.
AUTHOR: Hiraoka L R; Harrington J J; Gerhard D S; Lieber M R; Hsieh C L
CORPORATE SOURCE: Department of Pathology, Stanford University School of Medicine, California 94305-5324, USA.
CONTRACT NUMBER: 5T32CA09302 (NCI)
T32CA09151 (NCI)

T32CA09151 (NCI)
SOURCE: GENOMICS, **(1995 Jan 1)** 25 (1) 220-5.
Journal code: GEN. ISSN: 0888-7543.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L37374
ENTRY MONTH: 199509
AB We recently purified and cloned the **gene** for a **DNA** structure-specific endonuclease, **FEN-1**, from murine cells. The murine protein recognizes 5' **DNA** flap structures that have been proposed in **DNA** replication, repair, and recombination. Here, we report the sequence of the human FEN1 **gene** . The translated sequence is identical to peptide sequence obtained from maturation factor-1, which is 1 of the 10 essential proteins for cell-free **DNA** replication. The human protein has the same structure-specific **DNA** endonuclease activity as the murine protein. Two human chromosomal hybridization signals, 11q12 and 1p22.2, were observed by FISH analysis using human genomic clones homologous to the mouse **Fen-1 gene**. The localization on human 11q12 was confirmed using radiation-reduced hybrids. The mouse **Fen-1 gene** is assigned to chromosome 19 based on somatic cell hybrids. The significance of these FEN1 **gene** localizations in human and mouse is discussed.

L2 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1995:395079 CAPLUS
DOCUMENT NUMBER: 122:285186
TITLE: The characterization of the **FEN-1** family of structure-specific endonucleases: implications for **DNA** replication, recombination, and repair
AUTHOR(S): Harrington, John Joseph
CORPORATE SOURCE: Stanford Univ., Stanford, CA, USA
SOURCE: **(1994)** 154 pp. Avail.: Univ. Microfilms Int., Order No. DA9429935
From: Diss. Abstr. Int. B 1995, 55(7), 2539
DOCUMENT TYPE: Dissertation
LANGUAGE: English
AB Unavailable

L2 ANSWER 8 OF 10 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 95011546 MEDLINE
DOCUMENT NUMBER: 95011546
TITLE: Functional domains within **FEN-1** and RAD2 define a family of structure-specific endonucleases: implications for nucleotide excision repair.
AUTHOR: Harrington J J; Lieber M R
CORPORATE SOURCE: Department of Pathology, Stanford University School of Medicine, California 94305-5324.
CONTRACT NUMBER: 5T32CA09302 (NCI)
SOURCE: GENES AND DEVELOPMENT, **(1994 Jun 1)** 8 (11) 1344-55.
Journal code: FN3. ISSN: 0890-9369.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L26320

OTHER SOURCE: GENBANK-L26320
ENTRY MONTH: 199501
AB Structure-specific nucleases catalyze critical reactions in **DNA** replication, recombination, and repair. Recently, a structure-specific endonuclease, **FEN-1**, has been purified and shown to cleave **DNA** flap structures. Here, we describe the cloning of the murine **FEN-1 gene**. The nucleotide sequence of **FEN-1** is highly homologous to the Saccharomyces cerevisiae genes YKL510 and RAD2. We show that YKL510 and a truncated RAD2 protein are also structure-specific endonucleases. The substrate specificity of the truncated RAD2 protein implicates branched **DNA** structures as important intermediates in nucleotide excision repair. The polarity of these branched **DNA** structures allows us to predict the placement of **DNA** scissions by RAD2 and RAD1/RAD10 in this reaction.

L2 ANSWER 9 OF 10 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 94178266 MEDLINE
DOCUMENT NUMBER: 94178266
TITLE: The characterization of a mammalian **DNA** structure-specific endonuclease.
AUTHOR: Harrington J J; Lieber M R
CORPORATE SOURCE: Department of Pathology, Stanford University School of Medicine, CA 94305-5324.
CONTRACT NUMBER: 5T32CA09302 (NCI)
SOURCE: EMBO JOURNAL, **(1994 Mar 1)** 13 (5) 1235-46. Journal code: EMB. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L26320
ENTRY MONTH: 199406
AB The repair of some types of **DNA** double-strand breaks is thought to proceed through **DNA** flap structure intermediates. A **DNA** flap is a bifurcated structure composed of double-stranded **DNA** and a displaced single-strand. To identify **DNA** flap cleaving activities in mammalian nuclear extracts, we created an assay utilizing a synthetic **DNA** flap substrate. This assay has allowed the first purification of a mammalian **DNA** structure-specific nuclease. The enzyme described here, flap endonuclease-1 (**FEN-1**), cleaves **DNA** flap strands that terminate with a 5' single-stranded end. As expected for an enzyme which functions in double-strand break repair flap resolution, **FEN-1** cleavage is flap strand-specific and independent of flap strand length. Furthermore, efficient flap cleavage requires the presence of the entire flap structure. Substrates missing one strand are not cleaved by **FEN-1**. Other branch structures, including Holliday junctions, are also not cleaved by **FEN-1**. In addition to endonuclease activity, **FEN-1** has a 5'-3' exonuclease activity which is specific for double-stranded **DNA**. The endo- and exonuclease activities of **FEN-1** are discussed in the context of **DNA** replication, recombination and repair.

L2 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 8
ACCESSION NUMBER: 1992:37812 CAPLUS
DOCUMENT NUMBER: 116:37812
TITLE: Resistance to fenarimol in Nectria haematococca var. cucurbitae

cucurbitae

AUTHOR(S): Kalamarakis, A. E.; De Waard, M. A.; Ziogas, B. N.; Georgopoulos, S. G.

CORPORATE SOURCE: Lab. Plant Pathol., Agric. Univ. Athens, Athens, 118 55, Greece

SOURCE: Pestic. Biochem. Physiol. (**1991**), 40(3), 212-20
CODEN: PCBPBS; ISSN: 0048-3575

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Genetic work with 51 fenarimol-selected strains of N. haematococca cucurbitae identified a polygenic system for resistance with at least nine chromosomal loci involved. The mutant genes, designated **fen-1** to fen-9, gave low levels of resistance to fenarimol and to three other C-14 demethylation-inhibiting (DMI) fungicides, namely triforine, imazalil, and triadimenol. Haploid strains carrying two fen mutations exhibit higher levels of resistance, indicating additivity of **gene** effects. All fen-mutations appear to be pleiotropic, having more or less adverse effects on growth, sporulation, spore germination, pathogenicity, and tolerance of somewhat high temps. Accumulation of fenarimol in resistant strains was lower than in the wild type, suggesting that fen mutations code for a common resistance mechanism based on a permeability barrier. Various inhibitors of energy generation increased the accumulation level, indicating that accumulation is energy dependent and may be the result of passive influx and energy-dependent efflux. Lower accumulation in resistant strains is probably the result of increased efflux, as has been found with other fungi. A double mutant carrying the mutations fen-7 and fen-9 showed lower accumulation of fenarimol than a strain carrying the fen-7 only, indicating additivity of effects in this regard also.

=> log

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